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Demographic Variation in Cancer in Relation to Industrial and Environmental Influence

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Mortality data (183,064 deaths in a 30-year period, 1940–1969) by sex and three ethnic groups (white excluding Spanish-surnamed, nonwhite, and Spanish-surnamed) in 15 regions within the city (Houston), grouped around the air pollution sample collection stations have been analyzed. Valid contrast studies were possible in only one region within the city for all three groups and in six regions for white excluding Spanish-surnamed and nonwhite. There is evidence that the environmental factors of exposure over time to air and industrial pollutants in Houston has had a demonstrable effect in increasing regional mortality from cancer of the respiratory tract as well as from all other diseases and conditions of the respiratory tract and heart disease.

This study points out the need for mutually sustained collaboration of effort of the scientific and industrial communities to redirect their attention and research efforts to the exploration of the carcinogenic potential of the microchemical environment.

Epidemiological investigations of the pattern of cancer mortality in Houston, Texas have been compared with patterns of industrial and ambient pollution and evidence of a relationship has been found.

Realization that there is a regional pattern to cancer mortality closely paralleled by heart disease and that both are strongly correlated to urban density with a 30-year lag suggested two lines of research (*1*). The first was to try to find out what factors were hidden under the broad term, "urban density," and second, if there was a regional pattern for the U.S., was there one also for the regions within a state or perhaps, even within a densely populated city. Previously published studies (*2–6*) have demonstrated regional differences in morbidity and in mortality within the state of Texas. This current study shows that there are differences by region within the city of Houston. This study measures within city differences of mortality by 15 stations composed of grouped census tracts around air pollution collection sample stations.

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Originally Houston was important as a railroad center, and in our large cancer incidence data base there are 800 railroad workers. Houston's most phenomenal growth in the 1940's and 1950's, however, was due to its industrial complex, which has at its heart the ship channel which gave direct access to the Gulf of Mexico, abundant natural resources in petroleum, natural gas, sulfur, lime, salt, and water. Since the product of one chemical plant is the raw material of another, this combination of available natural resources and manufacturing plants created on the banks of the ship channel one of the world's greatest concentrations of petrochemical industries, dependent as they are on the byproducts of oil refining. The metropolitan area alone has seven oil refineries, has been producing over 80,000 barrels of oil a day since the 1950's, with oil rigs within a few yards of Main Street. About 40% of every basic petrochemical produced in the United States comes from the Houston area, as does 60% of the nation's sulfur, 80% of the synthetic rubber (more than a billion of the 1.5 billion pounds of the U.S. capacity of butadiene, the basic raw material for synthetic rubber), 45% of the liquid hydrocarbons, 90% of the nation's mag-

nesium manufacturing and 10% of the U.S. total of chemical fertilizers. The world's largest sulfuric acid chemical plant, producing 2,000 tons per day, is on the Houston Ship Channel.

Paradoxically, Harris County, despite the industrial and commercial expansion in Houston, its principal city, has more cattle than any other county in Texas, raises 28% of the nation's rice within 100 miles of the city, and is one of the world's leading cotton producers and markets.

The corporate city has an area of 328 square miles. It stands on the coastal plains near the western part of the Gulf of Mexico. The topography of the city is almost completely flat. During the summer Houston has a nearly tropical climate with few frontal passages, high relative humidity and predominately convective rain showers. During the winter months the polar fronts make their longest and deepest penetrations, but this activity is slowed by warm, moist southeasterly winds coming in from the Gulf. This combination is responsible for the production of heavy rains and dense fogs, intermittently from the first of October to the first of May. The average annual rainfall is 45 in. uniformly distributed throughout the year. The annual average temperature is 70°F with a range from 56 to 83°F. The relative humidity averages 76%.

The land-sea temperature differential combines with the pressure gradient and pattern west of the Bermuda High to cause prevailing winds to be southeasterly. Frequent periods of stagnation of short duration occur when air over Houston has thermal stability in the vertical attributable to surface-based radiation-type inversion. It is estimated that inversion frequency as a percentage of total hours on an annual basis for Houston is 25% with a range from 20% during the spring to 35% during the fall.

The wind rosette for Houston for 1969 is reproduced in Figure 1. Comparison of this rosette with the seasonal rosettes showed a general consistency of wind movement during the four quarters over center Houston and the northwestern part of the city. There are secondary extensions toward the southwestern quadrants which are probably partly accounted for by the strong northerly and northeasterly winds of short duration after the passage of cold fronts during the winter months.

The greatest source of particulate producing industry is along and just north of the Houston Ship Channel from an area east of Galena Park to an area east of downtown (station 2). Thus the local wind pattern has an effect on the distribution of suspended particulates, and the prevailing southeasterly winds play a major role in extending the patterns of pollution toward the northwest and, in the three months of winter, toward the southwest. There are other industrial sources of pollution in some areas of the city which will be mentioned in their context. Since there is no zoning in Houston, it is not possible to distinguish residential from industrial areas with any certainty. Another major source of pollution which is more generalized is the automobile. Houston has a high

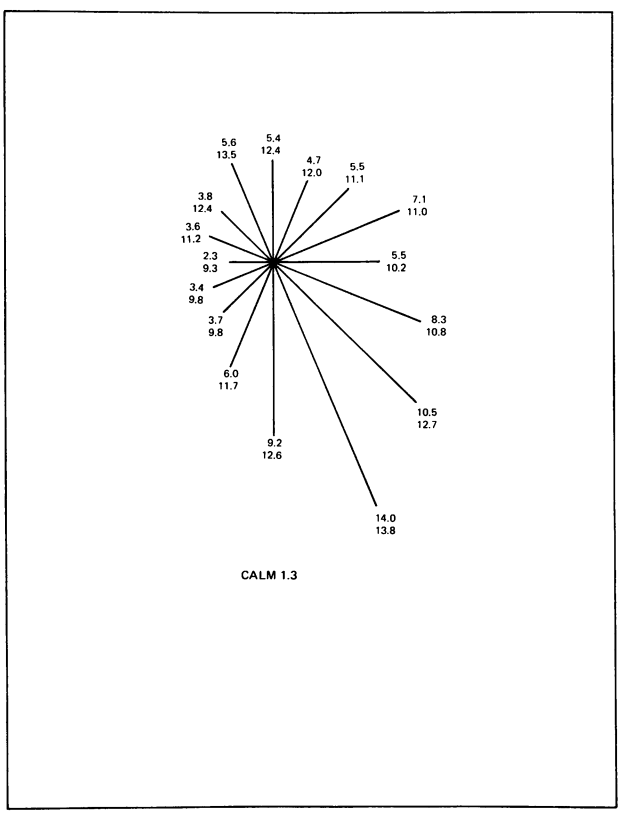

FIGURE 1. Annual wind rose, City of Houston (7). Directions are to the 16 point compass with north at the top. Upper number is the percent frequency of occurrence of wind from that direction. Lower number is the average speed from that direction in mph. Numbers are based on records collected during the period 1951 through 1960 at Houston International Airport.

dependency on the automobile second only to Los Angeles. There is a 113-mile highway system which completely circles the city, and the most heavily travelled highway runs through two of the stations under study (17 and 11). Approximately 178,000 cars daily, plus big commercial trucks, make this section of the highway the most used street in the city and one of the busiest in the nation.

Several surveys (*7, 8*) were conducted in 1967 and 1968 which give basic information by within city stations on the several settleable pollutants as well as those in the ambient air. Suspended and settleable particulates, including dustfall, gaseous air quality, sulfation levels, equivalent concentrations of ozone and aldehyde volume were measured. All measurements were based on 24-hr averages which obscure the concentrations that exist at times of peak traffic. Sawicki (*9*) showed seasonal variation in a national study in selected cities, with winter having 10–20 times the summer concentrations. Analysis by quarter in Houston revealed that the geographic distribution of suspended particulates followed the same pattern throughout the year except in July to September, when it dipped slightly, due possibly to a slowdown of production in the major industries during this period. The volume of particulate matter in the city is essentially constant through the year.

An east-to-west oriented pollution axis exists in Houston (Fig. 2). Since some emission control had been effected before the official pro-

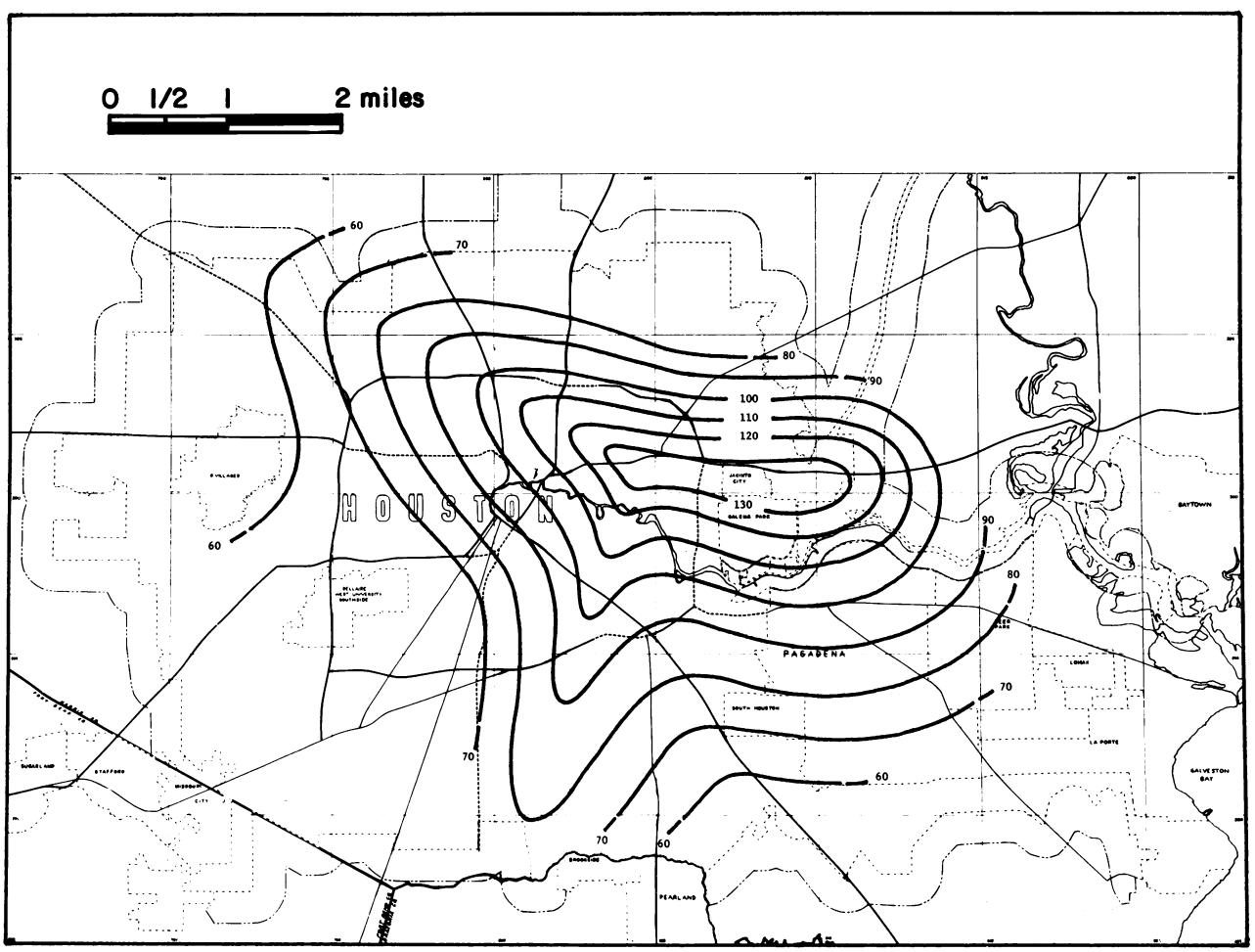

FIGURE 2. Annual distribution of suspended particulates ($\mu g/m^3$) for City of Houston (*8*): (——) highways and freeways; (- -) freeways proposed; (——) thoroughfares; (– · –) municipality limits; (— · —) Houston nuisance complaint limits; (▬) county line.

Table 1. Summary of emissions in City of Houston, 1967.[a]

Source category	Emissions, tons/yr				
	Sulfur oxides	Particulates	Carbon monoxide	Hydrocarbons	Nitrogen oxides
Transportation					
Road vehicles	1,990	3,260	439,200	56,530	24,650
Other	1,940	1,550	8,370	2,340	2,420
Combustion of fuels Stationary sources					
Industry	10	450	10	Neg.	5,350
Steam-electric	Neg.	40	Neg.	Neg.	900
Residential	10	130	10	Neg.	820
Commercial	Neg.	90	Neg.	Neg.	550
Refuse disposal					
Incineration	190	950	4,180	80	290
Open burning	—	720	3,830	230	500
Industrial process emissions	2,190	90,610	—	—	—
Grand total	6,330	97,800	455,600	59,180	35,480

[a] Data of City of Houston (*7*).

gram began, a reduction had already occurred, and the measurements reported in the 1967–1969 studies may reflect less exposure than occurred in the earlier years, of which the mortality rates reported in this study are a reflection. Table 1 gives the summary of emissions from these studies in 1967.

A more precise method of separating ozone measurements from the combination included under the term oxidants has given more precise readings of ozone levels. The collections are made in mobile units not in the regular collection stations. In Houston in the summer of 1975, for example, the ozone level reached five times the acceptable standard on one occasion. The sulfur dioxide survey showed that concentrations in some areas are high. Concentrations of nitrogen dioxide are evenly distributed throughout the city, which shows that motor vehicles are a main contributor. The aldehyde ambient air survey showed a large number of samples with concentrations above those known to cause eye irritation.

In January 1968, the accumulation of emission inventory data from individual industrial and commercial installations was started. A list of companies was compiled which included all known and potential, or even suspected, pollution sources. Throughout the Houston area, 155 major sources of pollution were identified. These sources can be classified as petroleum refining and petrochemical plants, cement plants, concrete ready mix plants, asphalt batch plants, paper pulp manufacturers, inorganic fertilizer operations, steel and steel alloy manufacturers, metal smelting and refining, sulfuric acid manufacturers, sandblasting and galvanizing. Among the types of pollutants emitted by industries included in the above list are particulate matter, both suspended and settleable, hydrocarbons, sulfur compounds, fluorides, nitrogen oxides, and carbon monoxide.

Houston has a network of 16 air pollution sampling stations strategically located throughout the city, reports from which have been available since 1968. Their location is indicated on the map showing their relationship to the highway network in and around the city (Fig. 3).

Current air pollution studies have the disadvantage of air sampling measurements occurring after the fact of mortality because no prior valid environmental data of 30 or 40 years ago exist.

Description of Houston

The population of Houston has tripled in the last 70 yr to approximately 1.2 million and doubled in the last 50 yr. The urban population of the city has doubled in 30 yr. During World War II immigration was massive, as much, for

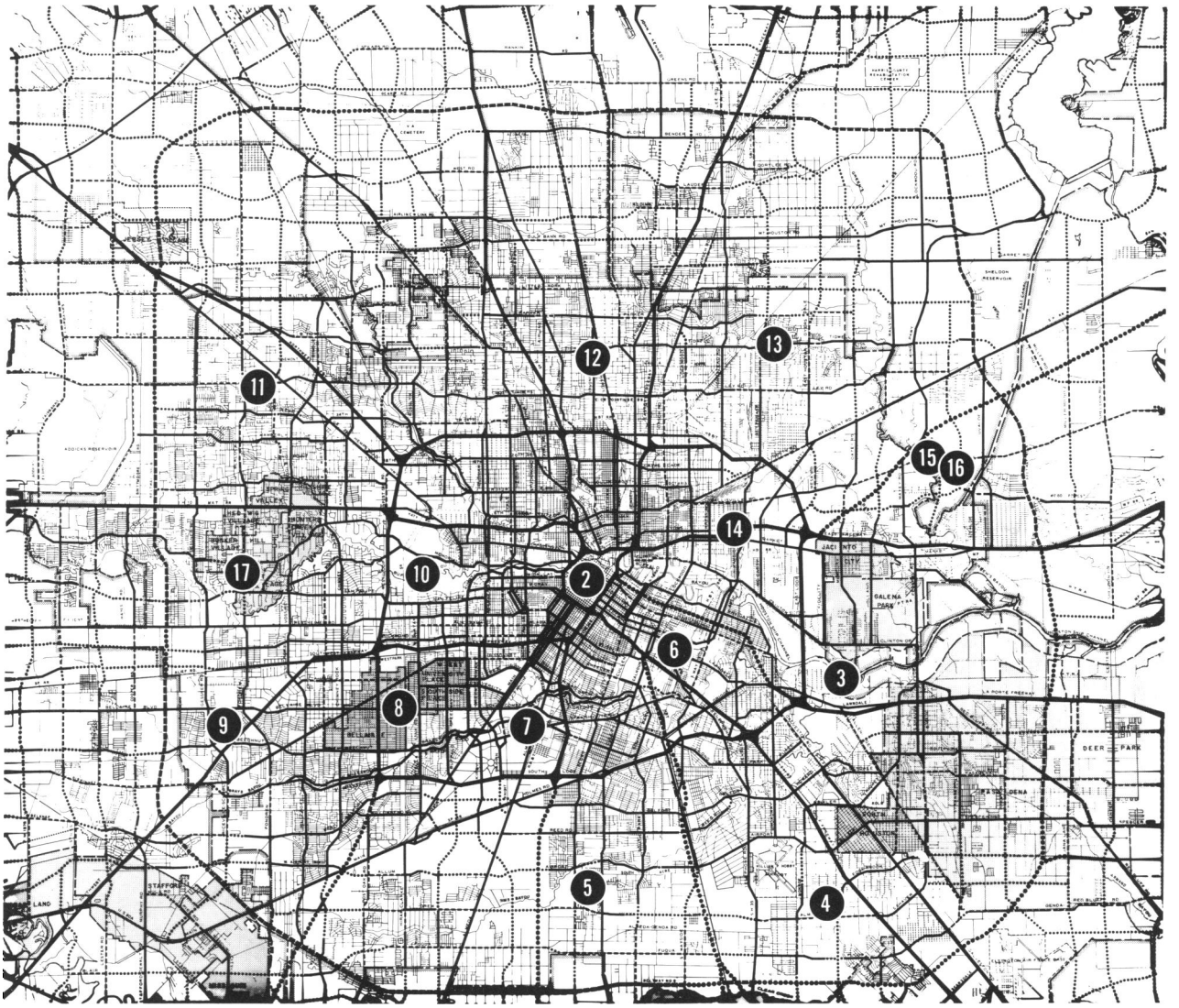

FIGURE 3. Major thoroughfares and streets with air pollution sample collection stations indicated. City Planning Department, Houston, Texas.

example, as 40,000 to the shipyards alone. The rest of Texas population growth is due to natural increase. In Houston, the bulk of the population increase is from Texans from other counties, and migrants from other states and countries. Houston's population differs from that of other urban centers in the United States in that it has proportionately fewer industrial craft workers, and more professional, technical, and white collar workers. The difference is due to the automation and technical nature of the major industries: processing oil, natural gas, and petrochemicals. The higher salaries paid by these industries have caused Houston to be a city of homes rather than apartments. Until the 70's there were very few highrise apartment buildings, for example, to create canyons which might alter wind flow and other ecological systems.

Methodology

In 1967, a four-year joint study was begun between the Department of Epidemiology of M. D. Anderson Hospital and the Department of Statistics of the Houston City Health Department. All death certificates since 1940 from all causes were recoded to the International Classification of Diseases 1955 rubrics, thus creating a homogeneous deck free from adjustments required by periodic changes in rubrics. The addresses were census-tracted. Census tracts covered by each of the air pollution sample collection stations were combined, age-specific and age-adjusted rates were calculated for the 30-yr period, for 5-yr averages, and for each year. Comparisons were then made between the stations for several causes of deaths, for total mortality, for demographic factors and for exposure to atmospheric pollutants. Knowledge of cigarette consumption by station is not available. The established urban smoking pattern can be assumed to exist in the city.

Demographic features of every station and the age-adjusted mortality rates by heart disease, cancer, and stroke as well as of all other causes and of total respiratory disease and of malignant respiratory disease were studied for each of the 15 air pollution collection sample stations. Slopes and rank correlation coefficients were calculated and studied. Significant differences were observed in mortality rates between the 15 stations in Houston for total respiratory diseases, for cancer of the respiratory tract, for cancer of all sites and also for heart disease and stroke. Demographic factors such as years of residence in the same house or the same census tract, the median age and age range, the ethnic composition and the median income were studied and compared.

Findings

During the period, 1940–1969, a total of 183,064 deaths from all causes occurred among residents in the city of Houston. The ethnic, sex distribution, by all causes, malignant respiratory, cancer, heart and stroke are shown in Table 2. Table 3 shows the total distribution by 5-yr groups for the period 1940–1969. Tables 4–6 give mortality rates for the 15 stations for 1965–1969. In this report, contrasts are made between the 1950–1954 year period and between the 1965–1969 year period by 15 stations within the city. The age-adjusted mortality rates for tuberculosis, influenza, bronchitis, upper respiratory diseases and other respiratory diseases are shown in Table 7 for white males.

Table 2. Age-adjusted mortality rates per 100,000 by sex and ethnic group: Houston, Texas (1940–1969).[a]

	All causes		Total cancer		Heart		Stroke		Total malig. respiratory	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
White										
Males	71,100	1014.9	11,499	167.0	28,836	439.1	5,909	96.3	3,438	48.2
Females	50,001	608.4	9,781	118.2	17,836	228.6	6,552	83.7	683	8.2
Nonwhite										
Males	28,367	1321.8	3,528	184.9	8,379	455.1	2,507	140.9	810	41.0
Females	23,303	1031.7	3,112	144.0	7,506	383.1	2,999	153.9	161	7.7
Spanish-surnamed										
Males	5,874	1342.7	625	184.2	1,388	460.6	341	114.0	148	46.3
Females	4,419	1072.8	614	183.4	970	356.6	321	110.7	45	14.6
Totals										
Males	105,341	1097.4	15,652	171.3	38,603	443.7	8,757	106.0	4,396	46.7
Females	77,723	715.3	13,507	125.8	26,312	263.6	9,872	98.6	889	8.4

[a] Adjusted to 1950 standard.

Table 3. Age-adjusted mortality rates per 100,000 by five-year averages for total population: Houston, Texas (1940–1969).[a]

Period	All causes		Total cancer		Heart		Stroke		Total malig. respiratory	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
1940–44	21,157	1253.7	2,170	131.1	6,500	428.5	1,914	127.6	183	11.1
1945–49	22,768	980.3	2,912	129.5	7,262	345.5	2,323	112.3	295	12.8
1950–54	25,906	860.2	4,077	139.1	8,753	319.0	2,702	100.4	566	19.0
1955–59	31,702	824.5	5,262	140.3	11,143	317.3	3,163	91.8	963	25.7
1960–64	37,764	836.5	6,568	148.0	14,630	351.1	4,053	99.5	1,348	30.2
1965–69	43,767	870.1	8,170	164.9	16,627	358.4	4,474	99.2	1,930	39.1
1940–69	183,064	894.6	29,159	146.2	64,915	348.1	18,629	102.1	5,285	26.5

[a] Adjusted to 1950 standard.

Table 4. Age-adjusted malignant respiratory mortality rates per 100,000 by sex and ethnic groups: Houston, Texas (1965–1969).[a]

Station	Males						Females					
	White		Nonwhite		Spanish-surnamed		White		Nonwhite		Spanish-surnamed	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
2	189	82.0	86	81.5	22	84.5	23	9.0	22	18.9	4	17.2
3	108	85.8					12	7.4				
4	60	56.3					16	13.8				
5	47	94.8	21	40.6			10	16.0	6	13.2		
6	108	101.3	34	53.5			17	12.8	10	13.2		
7	66	71.0	40	52.8			18	13.6	3	6.2		
8	104	71.6					44	20.8				
9	59	49.5					23	18.2				
10	85	50.7					24	10.5				
11	95	87.0					16	14.6				
12	181	78.7					35	13.4				
13	50	56.0	46	51.3			11	13.0	13	14.3		
14	24	69.5	16	69.4			5	12.6	3	13.9		
15/16	9	52.4					3	10.6				
17	28	47.4					17	20.4				
Total city	1213	71.5	305	57.6	55	72.1	274	13.3	64	12.0	19	27.0

[a] Rates given only when population base is sufficient. Adjusted to 1950 standard.

Table 5. Age-adjusted mortality rates per 100,000 total population by station: Houston, Texas (1965–1969).[a]

Station	Total all causes		Total heart		Total cancer		Total stroke		Total malig. respiratory		Total respiratory	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
2	8589	1112.07	3392	433.28	1395	180.88	870	111.03	346	44.89	932	120.20
3	2654	857.46	1002	347.52	500	162.41	262	94.48	132	42.21	302	95.82
4	1182	504.83	435	211.80	257	106.09	101	53.32	80	34.28	136	59.75
5	1868	819.94	630	338.89	362	165.59	184	102.77	87	42.05	174	82.90
6	3798	991.78	1438	393.19	648	169.63	398	111.68	174	45.48	398	103.07
7	3267	901.12	1206	327.74	520	143.51	370	99.75	128	35.58	328	91.55
8	2805	857.76	1214	381.63	681	198.36	287	96.78	149	41.63	260	75.73
9	1793	602.88	628	244.73	432	146.50	164	70.13	86	30.19	158	56.78
10	3065	808.57	1298	332.94	571	144.35	384	100.33	118	29.02	248	63.94
11	2636	858.63	966	365.52	556	181.32	267	108.12	135	46.05	257	88.64
12	5803	927.18	2189	388.22	1051	170.94	564	103.39	258	41.83	643	103.88
13	3724	932.66	1294	371.90	645	170.43	368	62.49	126	34.00	364	89.83
14	1261	1016.76	437	398.99	260	220.92	112	103.14	52	42.16	137	102.48
15/16	352	787.46	127	344.67	61	124.58	39	122.78	12	26.27	30	62.25
17	970	720.43	371	308.06	231	157.63	104	93.32	47	31.35	82	57.65
Total city	43,767	870.12	16,627	358.37	8170	164.93	4474	99.16	1930	39.06	4449	87.78

[a] Adjusted to 1950 standard.

Table 6. Age-adjusted mortality rates per 100,000 white males: Houston, Texas (1965–1969).[a]

Station	Malignant respiratory		All other respiratory excl. malig.		Total respiratory	
	No.	Rate	No.	Rate	No.	Rate
2	189	82.0	236	108.2	425	190.2
3	108	85.8	100	82.7	208	168.5
4	60	56.3	35	38.3	95	94.6
5	47	94.8	26	50.3	73	145.1
6	108	101.3	77	75.0	185	176.3
7	66	71.0	63	68.4	129	139.4
8	104	71.6	73	56.4	177	128.0
9	59	49.5	42	40.9	101	90.4
10	85	50.7	69	45.9	154	96.6
11	95	87.0	60	65.7	155	152.7
12	181	78.7	200	91.3	381	170.0
13	50	56.0	78	88.0	128	144.0
14	24	69.5	30	88.8	54	158.3
15/16	9	52.4	10	51.4	19	103.8
17	28	47.4	21	57.8	49	105.2
Total city	1213	71.5	1120	68.5	2333	140.0

[a] Rates adjusted to 1950 standard.

Table 7. Age-adjusted mortality rates per 100,000 white males for respiratory mortality excluding malignant respiratory: Houston, Texas (1965–1969).[a]

Station	Tuberculosis		Influenza		Pneumonia		Bronchitis		Other respiratory		Upper resp. infection	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
2	41	18.7	3	1.6	85	41.1	11	4.8	96	41.9	0	
3	13	10.8	0		43	36.1	6	4.7	38	31.1	0	
4	4	5.1	0		13	14.4	2	2.6	16	16.2	0	
5	1	1.5	0		7	11.4	3	2.6	15	34.7	0	
6	6	5.4	0		29	28.4	8	7.8	34	33.4	0	
7	7	8.4	0		26	28.7	4	4.5	26	26.9	0	
8	3	2.1	0		21	16.3	3	2.1	46	36.0	0	
9	1	.4	0		13	10.7	2	1.5	26	28.3	0	
10	9	5.5	0		20	14.6	3	1.6	37	24.2	0	
11	4	4.0	0		22	24.7	3	4.4	31	32.6	0	
12	17	7.9	1	0.6	69	27.4	18	9.7	95	45.6	0	
13	6	7.3	2	2.0	23	23.0	7	8.1	40	47.6	0	
14	8	20.9	0		9	29.3	1	3.0	12	35.7	0	
15/16	0				6	18.8	2	16.3	2	16.3	0	
17	2	1.7	0		9	23.4	0		10	32.6	0	
Total city	122	7.1	6	.4	395	23.1	73	4.7	524	33.3	0	

[a] Rates adjusted to 1950 standard.

In Houston, 70.9% of the population is white excluding Spanish-surnamed, 23.5% is nonwhite, and 5.6% is Spanish-surnamed. In this paper, the population group white excluding Spanish-surnamed will be referred to as white.

For total mortality for the total city, there are significant differences by sex in each group and between each ethnic group, with the male rates generally higher. When total cancer mortality is considered, rates for white males are higher than those for white females and rates for nonwhite males are higher than those for nonwhite females. There is no difference in the rates for total mortality between Spanish-surnamed males or females. White males, however, have significantly higher rates for total cancer mortality than either the Spanish-surnamed or the nonwhite, as do white females, generally. For total malignant respiratory diseases, the rates for males are higher than those for females, with the difference less pronounced among the Spanish-surnamed. White males have significantly higher rates than Spanish-surnamed males. The mortality pattern for heart disease is the same as that for cancer. Though there is no difference among males in the three ethnic groups in mortality rates for stroke, among females, the nonwhite have significantly higher mortality rates than the white.

It was hoped that ethnic differences could be studied by station within the city. Although the Spanish-surnamed and nonwhite are present in each station only in downtown Houston, station 2, is there enough representation from all three groups to make valid contrast studies. Total age-adjusted mortality rates from malignant respiratory diseases for males is 83.0/100,000. Males in all three ethnic groups have similar rates: white, 82.0; Spanish-surnamed, 84.1; and nonwhite, 81.5/100,000. Among females, the picture is quite different. The total rate for malignant respiratory diseases for females is 12.1/100,000. The rate for white females is significantly lower than that for nonwhite or Spanish-surnamed: white, 9.0; Spanish-surnamed, 17.2; and nonwhite, 18.9/100,000.

There are six stations within the city with large nonwhite and white populations. Downtown Houston, station 2, and the southeastern contiguous station 6, both fall in the high respiratory cancer mortality range for males. For station 2, the rates for white and nonwhite males are both high and nearly identical (white 82.0; nonwhite 81.5/100,000). For station 6, the white male rate is the highest in the city, 101.3/100,000. The rate for nonwhite males is 53.5/100,000, nearly half that for white. Station 5, which is contiguous to 6 in a south and southeasterly direction, has the second highest respiratory mortality rate for white males, 94.8/100,000, and a low rate for nonwhite males, 40.6/100,000. One potential explanation

is that the nonwhite males have moved in more recently. Station 13, northeast of center Houston and of the ship channel, has low and similar rates for white and nonwhite males, white 56.0 and nonwhite 51.3/100,000.

Station 14, with an intermediate rate range, borders the ship channel on the northeast upwind side and has nearly identical rates for white and nonwhite males; white, 69.5 and nonwhite 69.4/100,000. Station 7, southwest of center Houston, has an intermediate rate for white males, 71.0/100,000, and a low rate for nonwhite 52.8/100,000.

Rates for white and nonwhite females are similar except for two stations. In downtown Houston, station 2, the rate for white is half that for nonwhite females, 9.0 and 18.9/100,000. In station 7, nonwhite females, like nonwhite males in the same station, had half the rate of white females (6.2 and 13.6/100,000). On-site demographic studies should be made to ascertain the reasons for these differences. It is interesting that in one high mortality station (2), in one intermediate mortality station (14), and one low mortality station (13), white and nonwhite rates were nearly identical, which suggests that similar exposures tended to produce similar rates, regardless of ethnicity.

The age-adjusted mortality rates from lung cancer according to Mason (*10*) for the 20-year average from 1950–69 were for white males for the U.S. 37.9, for Texas 38.5, and for Harris County, 44.4/100,000. These rates in Texas are diluted by inclusion of the Spanish-surnamed, a generally low incidence group in this region. In Harris County, the rate for white males, excluding Spanish-surnamed, from 1950–54 was 33.2/100,000; fifteen years later the rate had more than doubled, to 68.3/100,000, in 1965–69. The phenomenon of doubling of the lung cancer rates had occurred in all but five of the fifteen stations in the city.

The diagrammatic map (Fig. 4) of the city shows that the high rates for white males are in stations contiguous to one another, that they follow the southeast-northwest wind gradient, that between the stations with low and high rates there are stations with rates intermediate between the low and the high. The stations in which the lung cancer rates have not doubled in the last 15 years all fall in the low lung cancer mortality belt except for one, which is in the intermediate rate range.

There are significant differences in rates from respiratory cancer for white males between the different stations. Among the white males, stations 2, 3, 5, 6, 11, and 12, have age-adjusted mortality rates for respiratory cancer above 79 per 100,000. The median age for white males in these stations ranges from 23.6 to 36.2, and between 74 and 81% have lived in the same house or the same area for over 5 yr, according to the census reports, and as long as 20 yr according to our verification of residence study. Stations 11 and 5 among white females are also high with rates of 14 and 16 per 100,000. There are in addition, three stations, 9, 17, and 8, with extremely high rates among females of 18.2, 20.4, and 20.8 per 100,000. Thus the effect along the southeast-northwest axis of pollution found in the air pollution surveys is reflected in abnormally high mortality rates from respiratory cancer. Where total cancer rates are high, heart disease mortality rates are also high. Heart disease mortality is often high where respiratory cancer is high, but mortality of heart disease was also high in stations 10, 13, 15, and 16, where the rates for respiratory cancer are lower than average.

In stations 7 and 8, white males have rates between 71 and 79 and in station 14, a rate of 69.5 per 100,000. The rates for the other 6 stations are between 47 and 56 per 100,000. In

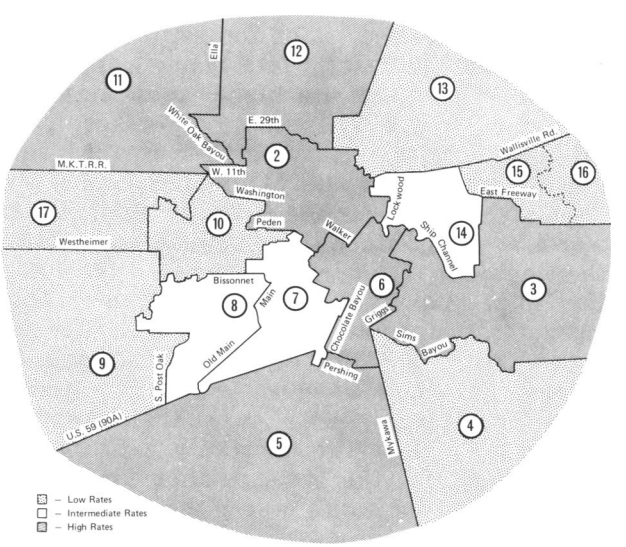

FIGURE 4. Air pollution sample collection stations, stations with high, intermediate, and low age-adjusted malignant respiratory mortality rates; 1965–1969, white males; Houston, Texas.

station 6, white males have a rate of 101.3 per 100,000 and in station 10, for example, half that rate, 50.7 per 100,000.

Station 6, an industrial area has a very high rate of respiratory cancer. In station 5 the median age is low, there is much heavy industry and the rates for respiratory cancer are high. It has also the fourth highest rate for white females.

Station 4 has many long-time established residential areas. It has a middle-range income, a median age of 25, and a low respiratory cancer rate. The stations with low rates are generally out of the path of the usual prevailing winds carrying pollution and have little or no industry within their borders. Presence or absence of atmospheric pollution seems to be the major difference between stations of high and stations of low respiratory cancer mortality. All ethnic groups follow the same pattern.

In center Houston (station 2), the most densely populated part of the city, the ethnic distribution in 1960 was white, 48.2%, Spanish-surnamed, 16.0%, and nonwhite, 35.8%. About three-fourths of the Spanish-surnamed and four-fifths of the nonwhite and white are long-time residents in the same house or census tract. When established cultural patterns are maintained for long periods of time, and the residence is the same, a true measure of the effect on mortality of the changing industrial and occupational exposures becomes available. In station 2, the Spanish-surnamed male respiratory cancer age-adjusted death rate was very high, 84.5 per 100,000. The Spanish-surnamed females in station 2 had a rate of 17.2 per 100,000, which is high for females.

The mortality rate from all causes and from heart, stroke, total respiratory diseases and respiratory cancer were ranked by each station in the city. Among the white population, station 2 ranks first in total mortality, second in heart disease, ninth in stroke, first in total respiratory diseases, but fifth in respiratory cancer mortality. Among females in station 2, the rank order is quite different. For total mortality, they rank sixth; for heart disease, first; for stroke, tenth; for all respiratory disease, sixth; and for respiratory cancer, twelfth.

Nonwhite mortality rates are high in all causes of death in station 2. The Spanish-surnamed in this region present a more mixed picture.

Station 8, 98.6% white in 1960, second highest in total mortality among males and third among females, contains Rice University and environs; it is largely residential and extends a great distance into a mixed small business and residential area. The median income is high. The respiratory cancer mortality rate is first among females but seventh among males. Total cancer mortality ranks first among males and females. Heart disease among males is the highest in the city in this station, with females third.

The stations are of special interest where the rank of total mortality differs greatly from the rank of respiratory cancer mortality. In station 3, for example, among white males, total mortality ranks eleventh, heart mortality, ninth, but respiratory mortality, third. The population in this station is 80.3% white.

Station 5, nearly half white and half nonwhite, with only 1.0% Spanish-surnamed, shows for white males, total mortality ranking thirteenth; heart mortality, thirteenth; and cancer of the respiratory system mortality, second. Among white females in station 5, total mortality is twelfth; heart, eleventh; and cancer of the respiratory system ranks fourth.

Station 4, 97.4% white in 1960, is fifteenth in total cancer for both white males and females. For males, it is tenth in rank for respiratory cancer mortality and for females, sixth.

The continuing analysis of the 30-year study by station is investigating the mortality rates for cancers of the stomach, pancreas, liver and bladder, and the leukemias, together with infections and allergies, the more subtle and difficult-to-measure manifestations of environmental contamination. The mortality rates for white males and females for influenza, pneumonia, bronchitis, emphysema, allergies and cancer of the respiratory tract for the last 30 years follow the same general trend for stations where the cancer respiratory mortality is high, other conditions are high, and the converse is true.

There was no consistent cause-and-effect relationship between the 1968–1969 readings of the several pollutants by station and the mortality rates for 1965–1969, but it must be remembered that there is a time lag between exposure and onset of cancer and other chronic diseases. Our mortality figures are a reflection of exposures from 10–30 years ago. The mortality rates from 1980–2000 will reflect the results of exposures

Table 8. Industries in Houston, Texas by major classification, 1972.[a]

Industrial classification	Stations															
	2	3	4	5	6	7	8	9	10	11	12	13	14	15—16	17	Total
Ordnance and accessories (19)	—	—	—	—	—	—	—	—	2	—	—	—	—	—	—	2
Food and kindred products (20)	33	15	3	—	9	5	8	5	10	9	9	3	8	1	—	118
Textile mill products (22)	4	1	1	—	1	—	—	—	—	2	2	—	—	—	—	11
Apparel and other finished products made from fabrics and similar materials (23)	24	2	4	—	5	6	1	2	9	2	2	2	4	1	—	64
Lumber and wood products, except furniture (24)	9	8	6	—	9	1	2	2	7	12	6	4	1	2	—	69
Furniture and fixtures (25)	22	9	10	1	8	7	1	2	5	9	4	2	5	2	—	87
Paper and allied products (26)	11	1	1	—	6	3	—	—	1	14	1	—	5	1	—	44
Printing, publishing and allied industries (27)	109	9	6	2	7	16	15	14	53	11	14	3	6	1	—	266
Chemicals and allied products (28)	39	17	9	14	15	6	—	6	13	36	2	8	16	3	—	184
Petroleum refining and related industries (29)	5	2	1	—	1	—	—	1	—	2	—	1	2	—	—	15
Rubber and miscellaneous plastics products (30)	24	6	13	4	13	5	—	5	4	15	7	3	5	—	—	104
Leather and leather products (31)	2	—	—	—	—	—	3	—	—	1	—	—	—	—	—	6
Stone, clay, glass and concrete products (32)	32	10	6	1	5	9	—	11	2	13	7	6	7	2	1	112
Primary metal industries (33)	20	8	3	5	14	1	—	4	1	10	5	5	8	4	1	89
Fabricated metal products, except ordnance, machinery and transportation equipment (34)	101	44	26	17	46	24	6	29	34	96	30	18	41	11	1	524
Machinery, except electrical (35)	84	43	40	9	35	27	3	27	26	59	22	9	18	6	—	408
Electrical machinery, equipment and supplies (36)	29	6	10	6	13	6	2	22	17	28	11	1	2	1	—	154
Transportation equipment (37)	5	6	1	1	3	1	—	1	1	6	9	4	3	2	—	43
Professional, scientific, and controlling instruments; photographic and optical goods; watches and clocks (38)	10	—	5	—	5	5	1	15	15	8	1	1	—	—	—	66
Miscellaneous manufacturing industries (39)	34	5	2	—	16	6	—	8	10	8	1	2	2	—	—	94
Total	597	192	147	60	211	128	42	154	210	341	133	72	133	37	3	2460

[a] Classification from Manufacturers' Directory (*11*).

being recorded today. How much of the mortality from other respiratory diseases is because of the reduction in the ability of the lungs to fight off respiratory infections after repeated long-term exposure to ozone and other pollutants, for example, even though the direct effects are not known to be dramatic, is problematical.

Significantly different rates from station to station exist within the city, even after age-ethnic adjustments. In addition to the higher mortality along the wind gradient, there is a general, though not completely consistent, relationship between the number of industrial installations and high respiratory mortality rates in certain stations. The major industry classifications are shown by station in Table 8 and Figure 5. In station 11, for example, numerous

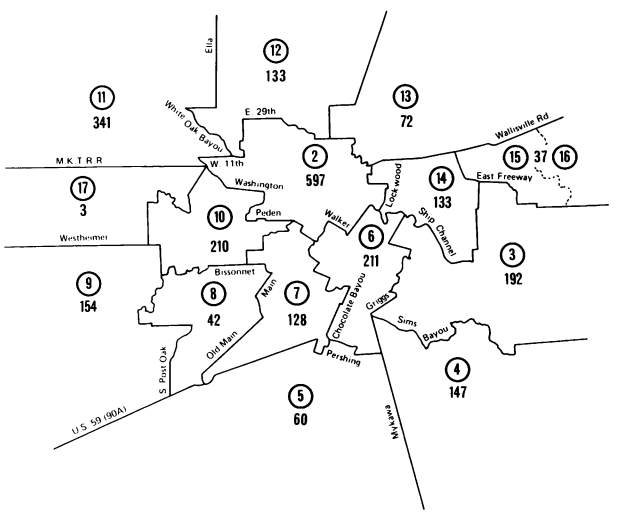

FIGURE 5. Major industries by stations from Manufacturers' Directory, 1972 (*11*). Circled numbers are station numbers.

industrial concerns that fall in the potentially carcinogenic category are present in a community which also lies in the direct path of the pollution-bearing wind. Although the ship channel industries are 10 miles from this station, the recently reported photochemical air pollution studies in the New York–New England area (*12*) demonstrate that ozone, for example, could be transported by the wind from the New York area across Connecticut to the Boston area (300 km) in 6–8 hr, and the same thing could be happening in Houston.

There is one anomalous station, 14, on the ship channel, where the rates are in the intermediate range and where the industrial complex has been concentrated for many years. The population is nearly equally divided, 48% nonwhite, 48% white. It was characterized by an above average rate in 1950–54 which did not double by 1965–69. This station is immediately upwind of the ship channel but lies in the path of more remote industrial pollution from eastern Harris County.

The evidence is overwhelming that the environmental factors of exposure over time to air and industrial pollutants in Houston has a demonstrable effect in increasing regional mortality from cancer of the respiratory tract as well as from all other diseases and conditions of the respiratory tract and from heart disease. Studies like this and demographic studies in general, provide clues relatives to other areas and population subgroups requiring further investigation into specific environmental factors.

It is apparent that there is a large area of scientific unknowns relative to the long-term effects from the complex microchemical environment. The carcinogenic potential of the chemical combinations that do exist in the community and work environment constitute these scientific unknowns. What is increasingly clear is that there is no simplistic answer to the cause of respiratory cancer or of cancer of many other sites.

This study points out the need for the scientific community to redirect its attention and research efforts to explore the carcinogenic potential of the microchemical environment—a virtually unexplored area.

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